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Case Report

Aortic Dissection: The Insidious Menace

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ABSTRACT

INTRODUCTION: Aortic dissection represents a critical emergency characterized by the cleavage of aortic wall layers, creating a deceptive false lumen. Associated with high mortality rates, prompt diagnosis and intervention are needed to ensure survival.

CASE: A 63-year-old male with no significant medical history presented to the emergency with two days of worsening mid-sternal chest pain. Upon presentation, he was hemodynamically stable. Blood pressure was 130/85 mm Hg (similar in both arms), heart rate 90 bpm, and saturating 98 % on room air. Physical examination did not reveal any new murmurs. EKG showed normal sinus rhythm, no ischemic changes, and a negative troponin50), but given low clinical suspicion for a pulmonary embolism, a CT angiography (CTA) was not pursued, especially since Wells Score was 3. The patient did meet one criterion (chest pain) for The Aortic Dissection Detection Risk Score, but unfortunately, neither a D-Dimer nor CTA was performed due to low clinical suspicion. Chest pain improved after Aspirin, sublingual Nitroglycerin, and Morphine. Troponin was negative x 2. A transthoracic echocardiogram, conducted after a delay of ten hours, disclosed a dilated aortic root measuring 4.7 cm and a 5 cm dilated ascending aorta. Additionally, a linear echo-density exhibiting flow, corroborated by color Doppler, raised suspicion of aortic dissection originating close to the right coronary cusp. Cardiothoracic surgery was immediately consulted, and a CTA was ordered. Regrettably, twenty minutes later, the patient became unresponsive with PEA cardiac arrest, and CPR was initiated. After 18 minutes of CPR, there was no return of spontaneous circulation, and the patient was pronounced deceased.

DISCUSSION: Our case underscores the deceptive nature of aortic dissection, which can present with benign physical examination, labs, and radiographic findings. In cases of uncertainty, prompt imaging with transthoracic/ transesophageal echocardiography or CTA should be performed as early detection and treatment can significantly improve prognosis.

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INTRODUCTION

Aortic dissection represents a critical emergency characterized by the cleavage of aortic wall layers, creating a deceptive false lumen. The mortality associated with ascending aortic aneurysm dissections increases by 1% per hour, for the first 48 hours, ultimately reaching about 50% if left untreated[1]. Furthermore, the mortality from aortic dissection remains at least 30% after arrival to the emergency department (ED), as a significant number of cases are missed by the providers[2]. Because of these high mortality rates, prompt diagnosis and intervention are needed to optimize survival. The following case demonstrates the severity an aortic dissection and highlights the strategies for urgent management.

CASE

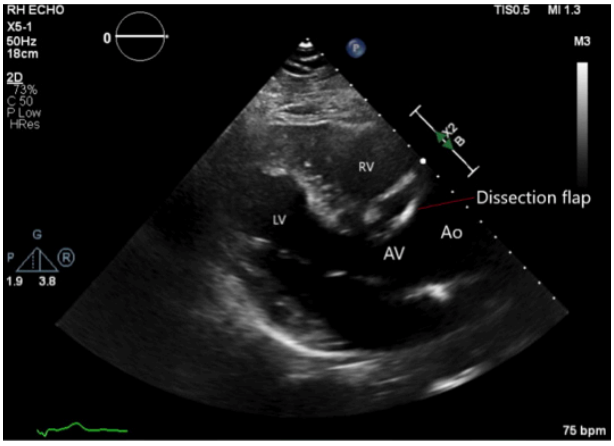
A 63-year-old male, with a medical history of tobacco and alcohol use who had not seen a medical provider in 20 years, presented to the emergency with mid-sternal chest pain/pressure. The pain began abruptly around 48 hours prior to arrival to the ED and had been gradually worsening. His discomfort radiated to his neck with associated shortness of breath and fatigue. He denied history of similar symptoms and denied alleviating or worsening factors. Vitals at presentation were blood pressure of 130/85 mm Hg (similar in both arms), heart rate 90 bpm, and saturating 98% on room air. Physical examination was grossly unremarkable, without evidence of any new murmurs. EKG showed normal sinus rhythm at

rate of 75 beats per minute with no ischemic changes. Lab work revealed troponin within normal limits and hemoglobin of 14.0 g/dL. Chest X-ray showed no significant abnormalities. Given low clinical suspicion for pulmonary embolism, a CT angiography (CTA) was not pursued, especially since Wells Score was 3. The patient did meet one criterion (chest pain) for The Aortic Dissection Detection Risk Score, but unfortunately, neither a D-Dimer nor CTA was performed due to the low clinical suspicion. Chest pain improved after aspirin, sublingual nitroglycerin, and IV morphine. Troponin levels at presentation and after five hours were within normal limits (reference range <0.03 ng/mL). A transthoracic echocardiogram (TTE) performed following hospitalization disclosed a dilated aortic root and ascending aorta with a linear echo-density exhibiting flow suggestive of aortic dissection originating close to the right coronary cusp. Cardiothoracic surgery was immediately consulted, and a CTA was ordered. However, the patient became unresponsive shortly thereafter with PEA arrest. Cardiopulmonary resuscitation (CPR) was initiated. After 18 minutes of starting CPR, there was no return of spontaneous circulation, and the patient was pronounced deceased.

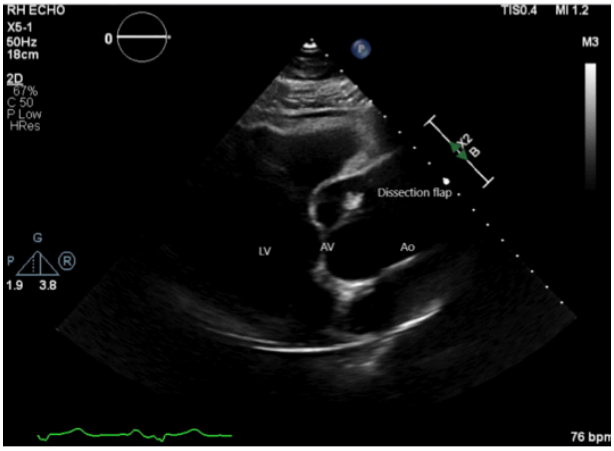
DISCUSSION

Acute aortic dissections (AADs) are uncommon, with an estimated range of occurrence of 2.6-3.5 per 100,000 person years[3]. Major risk factors include male sex, older age (mean age of incidence 63 years in males, 67 years in females), hypertension, smoking, cocaine use, and connective tissue disorders such as Ehlers-Danlos and Marfan syndromes. Interestingly, dissections occur more often during early morning hours and in winter months[4, 5]. The Stanford convention groups aortic dissections into type A (ascending intimal tear) and type B (descending intimal tear). AADs begin when the tunica intima tears and blood fills the tunica media, separating the intima from the media and forming a false lumen. AADs exhibit their mortality through a variety of mechanisms. The dissection may spread proximally to involve the aortic valve and leak into the pericardial space causing tamponade. It may also propagate distally to involve branch vessels, leading to ischemia of coronary, carotid, spinal, extremity, or visceral arteries. If the dissection is prominent enough, it may result in aortic rupture that is rapidly fatal. On presentation, pain is the most common symptom, occurring in >90% of patients, with 85% reporting that pain is abrupt in onset[6]. Depending on the type of dissection, pain may be reported as sharp/tearing, often located in the chest

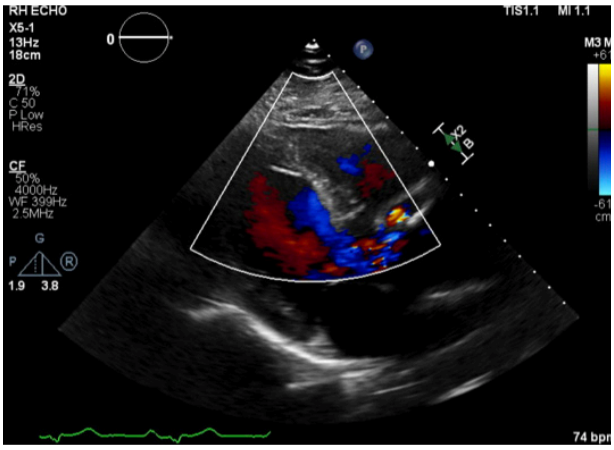
Figure 1. Transthoracic echocardiographic findings of aortic dissection.



A. Parasternal long axis view



B. Parasternal long axis view focusing on the aortic valve



C. Color doppler view of the aortic dissection

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or back, occasionally with radiation to the abdomen. Blood pressure deficit of >20 mmHg between the arms is common, occurring in up to 30% of dissections, and reflect an increased mortality[7]. A new diastolic murmur from aortic regurgitation occurs in 50-66% of ascending dissections[8]. Other less common findings are focal neurological deficits, syncope, and hypotension. Several scoring systems exist to stratify the risk and mortality of those with a possible AAD. The Aortic Dissection Detection Risk Score (ADD-RS) is the most common and considers three high-risk factors: associated conditions, pain features, and exam findings to guide practitioners in their workup. If a patient scores ⅓, recommendations are to proceed to D-dimer testing. If they score ≥ ⅔, recommendations are to proceed directly to CTA chest or other imaging[9].

EKGs are routinely ordered for chest pain, but rarely show evidence of AAD, even in the event of right coronary artery involvement[10]. Chest X-rays are helpful, which reveal a widened mediastinum in 60-90% of cases[11], although they have a lower sensitivity for type B dissections. Similar to the evaluation for pulmonary embolism, a D-dimer study is effective, especially for ruling out AAD. A D-dimer level of <500 ng/mL may rule out dissection, with a negative predictive value of 97.3%[12, 13]. Trials are ongoing to determine the effectiveness of new screening tests, including measuring the levels of smooth muscle myosin heavy chain and sST2 (an inflammatory peptide that is released during mechanical overload of the myocardium), which preliminarily have been shown to have similar and possibly superior sensitivities and specificities compared to conventional methods [14, 15]. Once suspicion is raised, the physician should proceed to cardiovascular imaging, which requires visualization of a dissection flap separating the false and true lumens to confirm the diagnosis. Common imaging modalities include MR/CT angiography and transthoracic/transesophageal echocardiogram (TTE/TEE), with the former typically reserved for hemodynamically stable patients and the latter selected for hemodynamically unstable patients[16]. In treating AAD, one must first stratify ascending (type A) vs descending (type B) dissections, as the former is a cardiothoracic surgical emergency, and the latter is managed medically if hemodynamically stable and without ischemic complications. In our patient, an ascending aneurysm was identified, but the patient rapidly deteriorated before CT surgery could be performed. Medical management, in the event of type B dissections or if CT surgery is unavailable for type A dissections, should prioritize maintaining a heart rate <60 bpm and systolic blood pressure between 100-120 mmHg. Beta-blockers (either esmolol or labetalol) are first-line agents to reduce shear force, followed by nitroprusside or nicardipine infusions if BP remains above 120 mmHg[17].

Our case discusses an uncommon presentation of AAD in which the initial workup—physical exam, labs, and radiographic findings—was benign. Deceptive presentations are not uncommon, as one autopsy study of 388 patients over 60 years found that 63% receiving medical care were not diagnosed with AAD until post-mortem[18]. In assessing high-risk clinical features of dissection, the only prominent feature that the patient in our case had was abrupt onset of mid-sternal pain, resulting in a low clinical suspicion for AAD. In fact, using the ADD-RS stratification, our patient only scored a ⅓. However, a ⅓ meets criteria for D-dimer testing, but no such testing or dedicated aortic imaging study was ordered. The diagnosis AAD was missed until the TTE was performed several hours after admission. Unfortunately, the patient rapidly converted to PEA arrest before any medical therapy or surgical intervention could be accomplished.

CONCLUSION

Acute aortic dissection (AAD) is a relatively uncommon but potentially devastating illness and therefore, requires a high index of suspicion among practitioners to avoid missed or delayed diagnosis. Our case presents a deceptive presentation of an AAD in which initial screening and definitive imaging assays were delayed, and the patient ultimately died from complications of AAD. Despite extensive literature on AAD, clinical suspicion for dissection is often suboptimal, and many cases are missed in the emergency department[2]. This case serves as a reminder to keep a high index of suspicion for dissection and to follow ADD-RS stratification for screening for AAD. At least one of the following observations can be identified in 96% of AADs: abrupt onset sharp/ripping thoraco-abdominal pain, pulse/blood pressure variation between extremities, and widened mediastinum on chest X-ray[19]. If present, these findings should prompt a swift investigation of aortic aneurysm. A D-dimer level < 500 ng/mL has a 97.3% negative predictive value for ruling out aortic dissection[20]; thus, in patients presenting with acute chest pain, obtaining a D-dimer along with serial troponins may be a reasonable strategy in the appropriate clinical setting, not to miss potentially life-threatening pathologies such as aortic dissection.

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